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THE HEMODYNAMIC BASIS
OF ATHEROSCLEROSIS.
FURTHER OBSERVATIONS:
THE LINEAR LESION.*

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A THEROSCLEROSIS should be considered as the reactive biological response of blood vessels to the forces generated by the flowing blood.¹⁻⁵ Flowing blood possesses kinetic energy which is expended on the walls of blood vessels as a compression (push) force,⁶ as a tensile (pull) force, or as a shear stress.⁷ Pressures are normal stresses (σ) acting perpendicularly to the surface. The effect of shear stress (τ) which acts parallel to the surface results in either a tensile or a compressional force in the vessel wall, depending on the magnitude and direction of the shear stress. In contrast to steady flow, which characteristically produces a relatively constant compres-

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sional or tensile stress at a given site, pulsatile flow is characterized by alternating compressional and tensile forces at the same site. The net effect of turbulent flow is also characterized by either compressional or tensile stresses occurring intermittently at a given site on the vessel wall.

MECHANISM FOR ATHEROSCLEROSIS

The hydraulic factors chiefly responsible for the development of atherosclerosis are the anatomical pattern (geometry of design)⁸⁻⁹ and the velocity profile in the blood vessel. In all flow patterns, however, a relative decrease in lateral pressure tends to develop at curvatures, branches, bifurcations, tapers, and zones of external attachment. These are sites of predilection for atherosclerosis.¹⁰⁻¹¹ The local decrease in lateral pressure is a tensile force, in effect a suction action, which exposes the endothelium to the lifting or pulling effect of the flowing blood. This is the initial stimulus. The initial biological response is a reparative or reactive thickening due to proliferation of intimal cells. With continuing blood flow the thickening intima encroaches upon the lumen as a plaque which progresses in size, shape, and pathological in situ change,^{12,13} depending on the varying pattern of blood flow and varying diminution in lateral pressure.

THE LINEAR LESION

Linear lesion is here described as a term applied to the intimal changes including atherosclerotic plaques which occur in portions of blood vessels which appear to be anatomically straight. Application of the laws of fluid mechanics and analysis of flow patterns in the thoracic aorta and in relatively straight portions of the coronary arteries reveal a rational and demonstrable basis for the localization, inception, and progressive development of linear lesions.

Linear lesions are here interpreted as the local biological or reparative response to hydraulic forces generated by the flowing blood. Although patterns of pulsatile blood flow in a straight vessel necessarily vary because of variations in velocity profiles and perturbations upstream due to anatomical design or geometry, idealized situations may be analyzed precisely and may serve as a basis for interpreting more complex patterns of blood flow.

The velocity profile for established streamline or laminar flow through a straight tube is shown diagrammatically in Figure 1a. It is notable that the velocity profile of flow is symmetrical and the velocity of flow increases from the wall toward the center or axial stream where it is maximal. When the

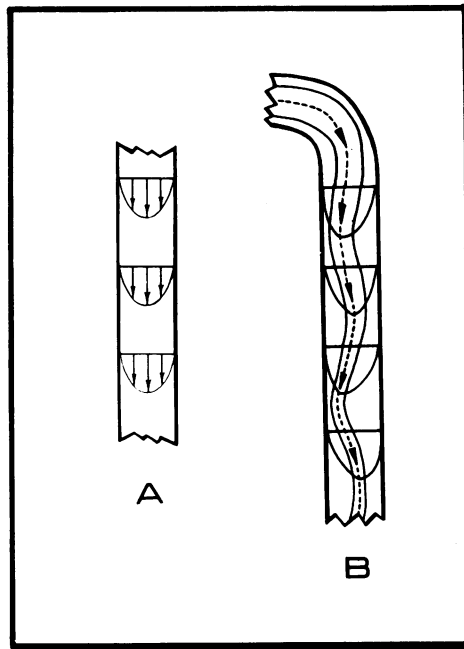


Fig. 1. Cross section of velocity profiles. Velocity distribution along a tube for laminar flow (A) and serpentine flow (B).

symmetrical velocity profile of fluid entering a section of straight tube is altered by a curvature or other perturbation, the axial stream or maximal stream line velocity is displaced from the midline and develops a serpentine path of flow. (Figure 1b) The velocity profile of serpentine flow presents a skewed distribution. The effect of the increased velocity¹⁴ and diminished lateral pressure¹⁵ adjacent to the boundary layer may provide the stimulus for the serpentine distribution of linear atherosclerotic plaques in relatively straight vessels.

The serpentine course of rivers and the effect of hydraulic forces on river banks provide geological counterparts to the hemodynamic basis of atherosclerosis with due regard to the differences in local conditions and specifications such as free surface flow, flow in curvatures, secondary flow, and the settling of particulate matter (Figure 2).

The serpentine pattern of flow is readily seen in a glass model which simulates the human aortic arch and thoracic aorta. (Figure 3) Autopsy specimens of human aortas (Figure 4) and coronary arteries (Figure 5) reveal linear atherosclerotic lesions.



Fig. 2. Rio Grande River showing serpentine course.



Fig. 4. Linear atherosclerotic plaques in thoracic aorta. White female, age 23. Note Ostial lesion downstream of origin of intercostal artery.

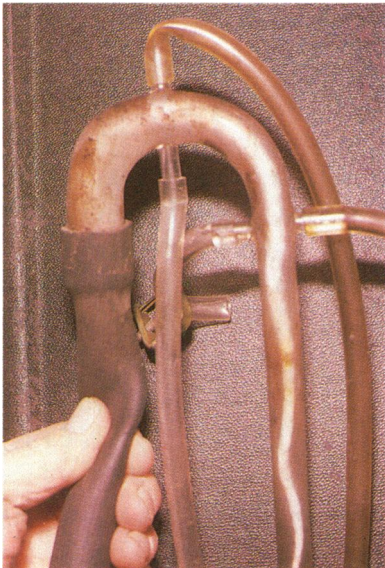


Fig. 3. Glass model simulating aortic arch and thoracic aorta showing serpentine course of air bubbles in axial stream (maximal velocity).



Fig. 5. Linear atherosclerotic plaques in coronary artery (L.A.D.). White female, age 15. Reproduced by permission from Texon, M.: The hemodynamic basis of coronary atherosclerosis with special reference to physical exercise. *Adv. Cardiol.* 18:122-35, 1976.

SUMMARY AND CONCLUSIONS

Application of the laws of fluid mechanics to the natural conditions of blood flow in relatively straight sections of blood vessels such as the thoracic aorta and segments of the coronary arteries strongly suggests a rational and demonstrable basis for the localization, inception, and progressive development of atherosclerosis as linear lesions.

Linear lesions are a result of a dominant serpentine flow with a skewed velocity profile that displaces the axial stream toward the wall. The increased velocity and diminished lateral pressure stimulate the endothelial layer to proliferate. Continuing blood flow leads to the progressive pathological changes which characterize atherosclerotic lesions.

This study of linear lesions further supports and compels the conclusion that atherosclerosis is a sequel of blood flow and that atherosclerosis represents the reactive biological response of blood vessels to the effects of the laws of fluid mechanics, namely, the forces (diminished lateral pressure) generated by the flowing blood at sites of predilection determined by local hydraulic conditions in the circulatory system.

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